

EXHIBIT 4

- 4a. Certificate for Hydroponics and
Appendix of Glossary pg.
- 4b. Certificate for Drug Program
- 4c. Articles on Environmental Tobacco Smoke
(ETS) or Second-Hand Smoke and Effects
- 4d. Sketch of FCI McKean, before and after
June 2004

EXHIBIT 4a

Certificate of Achievement

This certificate is awarded to

Demitrius Brown

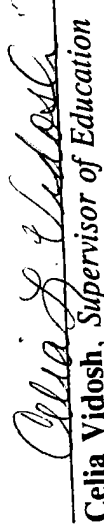
for opening your mind to the world of education
and completing

Hydroponics

Given this 17th day of June, 1998



Robert Hart, Hydroponics Instructor



Celia Vidosh, Supervisor of Education

H y d r o p o n i c • I n s t r u c t i o n a l • P a c k a g e

Appendix F : Glossary of Terms

Adhesion - The ability of water molecules to be attracted to certain materials.

Capillary Action - The ability of water to rise upwards in thin tubes of certain materials.

Chinampas - A word that describes the floating rafts used by the Aztecs to grow crops on.

Cohesion - The ability of water molecules to be attracted to one another.

Electroconductivity - The amount of electric current that dissolved solids can conduct in a solution.

Hydroponics - The art and science of growing plants in a nutrient solution with the roots supported in a medium other than soil.

Imbibition - The soaking of water into a porous material.

Macro Elements - Nine essential elements that plants require in large quantities to assure proper growth.

Micro Elements - Seven essential elements that plants require in small quantities to assure proper growth.

Monoecious Plant - A plant that possesses both pistillate and staminate flowers.

Parts Per Million (ppm) - Number of parts of a chemical found in one million parts of a particular gas, liquid, or solid.

Pathogen - An agent of disease.

Percent Germination - The percentage of seeds that are expected to germinate when planted.

EXHIBIT 4b

Certificate of Achievement



Demetrius Brown

21534-039

Successfully Completed

Drug Education Course

on September 23, 2003

Suzanne M. House

Suzanne M. House, Ph D; DAP
Coordinator

Amy Bankovic

Amy Bankovic, intern
Jessica Hayes, M.S. ED, CAC



EXHIBIT 4c



Secondhand Smoke

Today most people have heard the statement "secondhand smoke kills." But in society's increasing awareness of the health dangers of tobacco, of the lies manufactured by the tobacco industry, and of an emerging body of law supporting smokefree policies, it is not enough to simply state "secondhand smoke kills" without knowing how secondhand smoke is a health danger, whom it affects, where exposure is the most serious, and what can be done to stop it.

Throughout the years, the science of secondhand smoke has driven the secondhand smoke policy engine from separate smoking and nonsmoking sections to separately ventilated smoking rooms to 100% smokefree environments. We now know that 53,800 people die every year from secondhand smoke exposure. This number is based on the midpoint numbers for heart disease deaths (48,500), lung cancer deaths (3,000), and SIDS deaths (2,300) as calculated in the 1997 California EPA Report on Secondhand Smoke. And children are at significant risk to many acute and chronic diseases as a result of secondhand smoke exposure.

Since the 1986 Surgeon General's Report titled *The Health Consequences of Involuntary Smoking* stated that secondhand smoke can cause disease in nonsmokers, hundreds of studies have concluded not only this, but that exposure to secondhand smoke can result in death. Over the past 20 years, scientific research has become even more clear, resulting now in the ability to pinpoint the effects of secondhand smoke not just on particular organs, but on various ethnicities, types of workers, and socioeconomic classifications.

As the body of scientific evidence becomes larger and more precise, it is now possible to prove that smokefree policies not only work to protect nonsmokers from the death and disease caused by exposure to secondhand smoke, but also have an immediate effect on the public's health. On a larger scale, a study has confirmed that restaurants and bars located in smokefree cities have 82% less indoor air pollution than restaurants and bars in cities that do not have smokefree protection. Because of the mountain of evidence from these peer-reviewed, scientific studies, the Centers for Disease Control recently issued a warning for anyone at risk for heart disease to avoid smoke-filled indoor environments completely.

Secondhand smoke kills. Knowing the science behind it, as well as how smokefree policies protect the public from secondhand smoke, will help cement this in the minds of the public.

Americans for Nonsmokers' Rights
American Nonsmokers' Rights Foundation
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Medical & Other News

To print: Select File and then Print from your browser's menu

Title: **Research Shows Second-hand Smoke and High Cholesterol Damage Heart**

URL: <http://www.pslgroup.com/dg/6FA2.htm>

Doctor's Guide

March 26, 1996

SAN FRANCISCO March 26, 1996 -- The heart attacks that often kill non-smokers who are chronically exposed to second-hand tobacco smoke may result from a long disease process caused by smoke and high cholesterol acting together to damage cells lining artery walls, new experiments by UC San Francisco cardiologists suggest.

Damage done to these cells by smoke and fat may be offset somewhat by massive doses of a precursor of a molecule used by cells to send signals, or by vitamin E, the researchers found. They also determined that the damage may be made worse by the sex hormone testosterone. The researchers presented their preliminary findings today (March 25) at the annual meeting of the American College of Cardiology in Orlando, Fla.

Epidemiological studies comparing deaths among populations with differing lifestyles indicate that each year 50,000 or more non-smokers die from heart attacks attributable to second-hand smoke. Heart disease greatly outstrips lung cancer or other cancers as the most prominent, deadly health risk faced by non-smokers thus exposed. The new UCSF findings shed light on the biological reasons why second-hand smoke is harmful to heart health.

"Our experiments demonstrate that smoking and diet can affect the health of cells that are vital in preventing the clogging and hardening of arteries," says Stuart Hutchison, MD, a clinical instructor in cardiology at UCSF who presented some of the group's findings.

The UCSF group focuses on gaining a better understanding of how diet, smoking and hormones affect a microscopically thin layer of cells, called endothelial cells, which lines the inner surface of blood vessels.

By preventing blood-borne molecules from attaching to blood vessel walls, healthy endothelial cells prevent the build-up of plaque, Hutchison explains. The cells also release and respond to chemicals that cause blood vessels to widen and narrow as the physiological situation demands.

If these cells are lost or damaged, blood vessels are more likely to accumulate plaque, and the blood vessels become less capable of adjusting their diameters to meet the body's oxygen needs.

Plaque accumulation and its possible role in heart disease has been appreciated for centuries, Hutchison points out. In comparison, the loss of the ability to regulate blood vessel diameter has been known for

less than two decades, and research into its contributions to heart disease has intensified and become more fruitful in recent years, according to Hutchison.

The researchers used rabbits to perform controlled experiments that would be difficult or impossible to conduct in humans. On a small scale, the heart and blood vessel anatomy of rabbits mimics that of humans, and the circulatory systems of the two mammals are believed to respond similarly to life's insults.

The UCSF scientists exposed male rabbits to smoke and a high cholesterol diet for ten weeks. To conduct detailed investigations of endothelial cell performance and of affects on blood vessels, the researchers studied the responses of living slices of aorta bathed in a saline solution similar to blood, bubbling oxygen into solution to keep cells alive.

The UCSF group measured plaque build-up, as well as the ability of aortic slices from treated animals to contract and relax in response to pharmacological substances. Both measures of heart disease were adversely affected when rabbits were fed a high cholesterol diet and exposed to tobacco smoke.

The purpose of using L-arginine in the studies, Hutchison says, is that it is a building block used by endothelial cells to make nitric oxide, a signaling molecule in the cells. The cells produce nitric oxide to control the diameter of blood vessel cells.

The researchers found that nitric oxide production dropped in endothelial cells exposed to smoke. Supplementing the diet with L-arginine helped the aortic slices to remain flexible. This suggests that L-arginine leads to increased production of nitric oxide, causing the vessel to relax, Hutchison said.

The UCSF team also bathed some of the aortic slices from the rabbits in a solution containing physiologic concentrations of various sex hormones, including testosterone and estrogen. "In these male rabbits, estrogen did not improve the impaired response, and testosterone actually worsened it," Hutchison says. "This suggests that in male animals testosterone may participate in some aspects of atherosclerosis.

"This work raises questions about a possible role of sex hormones as protective or contributing factors in heart disease," Hutchison adds. "With more women trying to weigh the risks and benefits of post-menopausal estrogen therapy, and with many adolescent boys and men abusing male sex hormones in an effort to better compete athletically, understanding these connections becomes increasingly important."

In comparison to men, women are believed to be somewhat protected from heart disease by estrogen until production of the hormone drops at menopause.

The researchers used vitamin E, an anti-oxidant, to investigate the possible role of oxidation in damaging blood vessels and endothelial cells. They wanted to know if oxidation is implicated in causing a blood vessel to become less flexible.

Vitamin E restored the ability of blood vessels to relax in response to pharmacological agents in the smoke-exposed, high cholesterol rabbits, but research on aortic slices in this group has thus far been inconclusive, Hutchison says.

"These preliminary studies are helpful in understanding how cellular changes contribute to heart disease," Hutchison says. "However, we used massive doses of supplements to help us examine very specific aspects of blood vessel behavior and our results do not lead us to recommend dietary supplements for human heart disease at this time.

"Ultimately," Hutchison adds, "We hope similar studies will help us learn how to better detect and prevent these harmful biological events before a heart attack occurs."

Other UCSF Division of Cardiology physicians who participated in the research presented today include William Parmley, MD, professor and chief; Kanu Chatterjee, MD, professor; Stanton Glanz, MD, professor; Khrishna Sudhir, MD, assistant professor; Tony Chou, MD, assistant professor; and Prakash Deedwania, MD, a clinical professor of medicine at UCSF who is also the chief of cardiology at the Veterans Administration Hospital in Fresno.

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All He

Lung Cancer Can Strike Nonsmokers

Hea

Yahoo! Health

August 10, 2005 08:41:26 PM PST

By Amanda Gardner
HealthDay Reporter

WEDNESDAY, Aug. 10 (HealthDay News) — The news that Dana Reeve, the widow of "Superman" actor Christopher Reeve, has lung cancer proves the disease can — and does — strike anyone.

She is only 44 years old. And never smoked.

Her revelation Tuesday followed by two days the lung-cancer death of ABC news anchor Peter Jennings at age 62.

Jennings was both a smoker and an ex-smoker: He quit for 20 years but admitted starting again after the Sept. 11 terrorist attacks.

While Jennings' ordeal was probably due to cigarettes, Reeve's struggle is less understandable.

Some 85 percent to 90 percent of lung cancer cases occur in smokers, said Dr. Ruth Oratz, an associate professor of medicine at New York University School of Medicine and an advisor to the American Lung Association.

Still, that leaves some 20,000 to 25,000 people who will be diagnosed with lung cancer this year in the United States who never smoked, Oratz added.

Lung cancer's overall toll is huge — it's the most deadly cancer, killing more Americans than any other form of the disease, including ovarian and breast cancer combined. According to the American Cancer Society, there will be about 172,000 cases of lung cancer in the United States this year — 93,010 among men and 79,560 among women. And about 1

people will die of the disease – 90,490 men and 73,020 women.

But the face of lung cancer is changing and, sadly, Dana Reeve may represent that newer face.

"What we've been seeing in the last 10 to 15 years is a gradual change in the standard lung cancer patient," said Herman, a thoracic surgeon and lung cancer specialist who is chief of minimally invasive thoracic surgery at Long College Hospital in New York City.

For one thing, Reeve is a woman.

"Traditionally, in the past, lung cancer was primarily a male disease where the ratio was maybe 75 to 80 percent for the rest females," Herman said.

Now that's changing. "Lung cancer is increasing in incidence in women smokers and nonsmokers," Oratz concurs. "We know why that's happening in nonsmokers."

And lung cancer is striking younger people, Herman said.

These demographic shifts seem related to biological shifts.

In the past, lung cancers tended to be predominantly of the squamous cell subtype. This form of lung cancer was closely associated with smoking, Herman said.

In the last decade or so, the predominant cell type has become adenocarcinoma, which is less tied to smoking.

"Clearly, adenocarcinomas are much more likely to occur in smokers but a much larger percentage of people with adenocarcinoma may not be smokers," Herman said. "The fact is that adenocarcinoma is increasing in frequency. To squamous means you're going to get more nonsmokers with the disease."

Women have a slightly higher propensity to get this type of lung cancer. And, adenocarcinoma also tends to hit at a younger age because it's not so dependent on the build-up of years of irritation caused by smoking, he added.

And the younger the patient, the more aggressive the tumor, it seems.

"If lung cancer is discovered at a younger age, it seems to be a more aggressive type of cancer," said Dr. Paul Kvale of the American College of Chest Physicians and a pulmonologist at Henry Ford Hospital in Detroit. "Likewise with gender and family history, the two of those together seem to be associated with a more aggressive type of lung cancer."

But there are a number of other possible risk factors.

Passive smoking is a big one. "We think secondhand smoke is a very important risk factor, particularly if you live in a household where others smoke," Kvale said.

"Women who live with a partner who smokes or who work in a workplace that is smoky are at a major increased risk," Kvale said. "For some reason that is not well understood, women are at a higher risk than men for lung cancer given the exposure."

Exposure to asbestos is another risk factor, but one that is more pertinent to men because it is an occupational risk rather than a lifestyle risk factor.

Radon is another possible environmental risk factor, although quantifying how much of an additional risk has proven difficult, Kvale said. A radioactive gas, radon comes from the natural decay of uranium that is found in nearly all soils. It is

many homes and is known to cause lung cancer, according to U.S. health officials.

And a number of individuals who have received radiation treatment for other diseases in the past may be at increased lung cancer, Oratz said.

Genetics also play a small role in the development of lung cancer. "There's not a strong genetic linkage but if you family where other family members have lung cancer you will have a slight increased risk," Oratz said.

Or it could be a combination of factors. "When you couple several of those things, for example, secondhand-smoke and family history, then you've really got several things that are increasing your own risk," she said.

Then there's the unknown.

"Undoubtedly there are other issues that we haven't yet discovered," Kvale said.

More information

Visit the [American Lung Association](#) for more on lung cancer.

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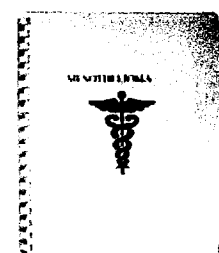
Lung Cancer Symptoms

- A cough that doesn't go away and gets worse over time
- Constant chest pain
- Coughing up blood
- Shortness of breath, wheezing, or hoarseness
- Repeated problems with pneumonia or bronchitis
- Swelling of the neck and face
- Loss of appetite or weight loss
- Fatigue

These lung cancer symptoms may be caused by lung cancer or by other conditions. It is important to check with a doctor.

To help find the cause of lung cancer symptoms, the doctor evaluates a person's medical history, smoking history, exposure to environmental and occupational substances such as asbestos, and family history of cancer. The doctor also performs a physical exam and may order a chest x-ray and other tests. If lung cancer is suspected, sputum cytology (the microscopic examination of cells obtained from a deep-cough sample of mucus in the lungs) is a simple test that may be useful in detecting lung cancer. To confirm the presence of lung cancer, the doctor must examine tissue from the lung. The removal of a small sample of tissue for examination under a microscope by a pathologist can show whether a person has lung cancer. A number of procedures may be used to obtain this tissue:

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- Bronchoscopy. The doctor puts a bronchoscope (a thin, lighted tube) into the mouth or nose and down through the windpipe to look into the breathing passages. Through this tube, the doctor can collect cells or small samples of tissue.
- Needle aspiration. A needle is inserted through the chest into the lung cancer tumor to remove a sample of tissue.
- Thoracentesis. Using a needle, the doctor removes a sample of the fluid that surrounds the lungs to check for cancer cells.
- Thoracoscopy. Surgery to open the chest is sometimes needed to diagnose lung cancer. This procedure is a major operation performed in a hospital.

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Symptoms of Mesothelioma: ☐ Yes ☒ No

Display symptoms

Lung Cancer: ☐ Yes ☒ No

Fluid in the Lungs/ Pleural ☐ Yes ☒ No

Effusion:

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Physicians *for a* Smoke-Free Canada

Health Effects of Chemicals found in Cigarette Smoke

Click on a chemical in the table below to view a short summary of its properties and health effects. A source list of studies on which this summary is based can be obtained by contacting or you can download the PDF version by clicking here.

AMMONIA	HYDROGEN CYANIDE
1,3-BUTADIENE	HYDROQUINONE
1- AMINONAPHTHALENE	ISOPRENE
2- AMINONAPHTHALENE	LEAD
3-AMINOBIIPHENYL	METHYL ETHYL KETONE
4-AMINOBIIPHENYL	NAT
ACETALDEHYDE	NICKEL
ACETONE	NICOTINE
ACROLEIN	NITRIC OXIDE
ACRYLONITRILE	NNK
BENZENE	NNN
BENZO[a]PYRENE	PHENOL
BUTYRALDEHYDE	PROPIONALDEHYDE
CADMIUM	PYRIDINE
CARBON MONOXIDE	QUINOLINE
CATECHOL	RESORCINOL
CHROMIUM	STYRENE
CRESOL	TAR
CROTONALDEHYDE	TOLUENE
FORMALDEHYDE	

AMMONIA

- Ammonia possesses a sharp, irritating odor detectable as low as 1 ppm.\
- At high concentrations, ammonia causes intense irritation, severe eye damage, and asthma.

top

2-AMINONAPHTHALENE

- 2-aminonaphthalene causes cancer in humans.
- There is no safe exposure limit for 2-aminonaphthalene.
- Absorption of 2-aminonaphthalene occurs both by inhalation and through the skin.
- Because it causes cancer, the industrial use of 2-aminonaphthalene is restricted or banned.

top

1-AMINONAPHTHALENE

- 1-aminonaphthalene has been shown to cause lung, liver and leukemia cancers in animals.
- 1-aminonaphthalene may cause cancer in humans.
- 1-aminonaphthalene has been shown to have moderate toxicity in fish.
- Absorption occurs both by inhalation and through the skin.
- Absorption through the skin may occur without a sense of irritation or other warning.
- The main industrial uses of 1-aminonaphthalene include dyes, rubber, and weed control.

top

4-AMINOBIIPHENYL

- 4-aminobiphenyl is confirmed to cause cancer in humans.
- The carcinogenic nature of 4-aminobiphenyl has been known since at least 1974.
- This chemical has been called 'one of the most potent known bladder carcinogens'.
- There is no known safe level of 4-aminobiphenyl.
- Absorption occurs through the skin.

- 4-aminobiphenyl is no longer produced on a commercial scale for use in industry.

top

3-AMINOBIIPHENYL

- 3-aminobiphenyl is a mutagen, and causes mutation in microorganisms.

top

BENZO[a]PYRENE

- Benzo[a]pyrene (B[a]P) is suspected to cause cancer in humans.
- There is a significant correlation between B[a]P exposure and lung cancer mortality.
- B[a]P was found to cause cancer in animals and fish in every study to date.
- Animal studies showed that as low a dose as 0.05 mg B[a]P caused tumors.
- B[a]P deposits in the lung. Elimination of B[a]P from the lung is severely restricted by cigarette smoking.
- Cancer is more likely to occur with repeated B[a]P exposures than with a single dose of the same amount.
- B[a]P exposure may also cause skin cancer, dermatitis, photoallergy, non-neoplastic respiratory disease and emphysema.
- Exposure to B[a]P results in decreased reproductive capacity for both males and females.
- Absorption of B[a]P also occurs through the skin.

top

FORMALDEHYDE

- Formaldehyde is suspected to cause cancer in humans.
- Formaldehyde occurs naturally at 0.12 to 0.38 parts per BILLION [ppb]. Sidestream smoke increases this by 0.23 to 0.27 parts per MILLION [ppm] (a 1000+ increase).
- Long-term exposure at levels greater than 0.1 ppm appears to be a risk for cancers of the lung, pharynx, buccal cavity, liver, bone, skin, prostate gland, bladder, kidney and eye, leukemia and Hodgkin's disease.
- Animal studies showed formaldehyde is an irritant to eyes, nose, throat and lungs, and causes cellular changes in the upper respiratory tract, a decrease in respiratory rate, and adversely affects the liver.
- Formaldehyde exposure greater than 0.22 ppm is linked to respiratory symptoms such as cough, phlegm, chronic

- bronchitis, asthma, shortness of breath and chest colds.
- Formaldehyde is known to produce allergic reactions and induction of asthma-like conditions, lightheadedness, dizziness, diminished dexterity, itching eyes, dry and sore throats, disturbed sleep, unusual thirst, and malignant disease in humans.
- Human eyes are sensitive to formaldehyde at concentrations of 0.01 ppm, and are irritated by formaldehyde at concentrations of 0.05 to 0.5 ppm.
- The main uses of formaldehyde in industry include fertilizer, dyes, disinfectants, germicides, preservatives, and embalming fluid.

top

ACETALDEHYDE

- Studies have shown that acetaldehyde causes cancer in animals, and may cause cancer in humans.
- Small amounts of acetaldehyde irritate the eyes, skin, and respiratory tract of humans and animals.
- Animal studies in which pregnant rats were exposed to acetaldehyde found that acetaldehyde interfered with the exchange of nutrients from the mother to the placenta, resulting in growth retardation, malformation, delayed bone growth and death of the fetus.
- Acetaldehyde may increase the absorption of the other hazardous chemicals in tobacco smoke into the bronchial tubes.
- The main industrial uses of acetaldehyde include silvering of mirrors, leather tanning, fuel, glue, dyes, plastics and synthetic rubbers.
- Acetaldehyde decomposition products include carbon monoxide.

top

ACETONE

- Acetone is an irritant to eyes, nose and throat.
- Acetone irritates, dries, and may burn skin.
- Absorption occurs both via inhalation and through the skin.
- Higher doses can cause dizziness, lightheadedness, damage to the liver and kidneys.

top

ACROLEIN

- Acrolein has not been found to cause cancer. However, in the body, acrolein produces glycidaldehyde which

does cause cancer.

- Long term inhalation studies on animals found that acrolein causes emphysema and inflammation of the lung, liver and kidney.
- Acrolein is intensely irritating to the eyes and upper respiratory tract in human and animals. Acrolein is 5 times stronger an irritant than formaldehyde, acetaldehyde or crotonaldehyde (all of which are found in tobacco smoke).
- The main industrial uses of acrolein include polyurethane manufacture, polyester resins, herbicides and tear gas.

top

PROPIONALDEHYDE

- Inhalation of propionaldehyde causes severe irritation of the respiratory system.
- Propionaldehyde causes irritation to skin and eyes.

top

CROTONALDEHYDE

- Crotonaldehyde is known to cause cancer in animals.
- Crotonaldehyde causes cancer by interfering with DNA function (a genotoxic carcinogen).
- Crotonaldehyde is a fast-acting (within seconds) irritant to the nose and upper respiratory tract.
- The main use of crotonaldehyde in industry is as a warning agent in fuel gases.

top

METHYL ETHYL KETONE

- Methyl ethyl ketone causes nose, throat, and eye irritation in humans at moderate levels.
- The odor of methyl ethyl ketone is detectable at 10 ppm.
- The main uses of methyl ethyl ketone in industry include solvents, resins, artificial leather, rubbers, lacquers, varnishes and glues.

top

BUTYRALDEHYDE

- Butyraldehyde is an irritant to eyes, nose, throat and lungs.
- Higher doses of butyraldehyde causes dizziness and

lightheadedness, and may burn skin.

- The main industrial uses of butyraldehyde include resins, solvents and plasticizers.

top

HYDROGEN CYANIDE

- Hydrogen cyanide causes nasal irritation, confusion, headache, dizziness, weakness and nausea in humans at moderate doses.
- At higher doses, hydrogen cyanide causes asthenia, vertigo, loss of weight and gastrointestinal problems.
- The main uses of hydrogen cyanide in industry include fumigation, as an insecticide, electroplating, metallurgy and photography.

top

NICKEL

- Inhalable, insoluble nickel is confirmed to cause cancer in humans.
- Up to 5% of the general population are sensitized (allergic) to nickel.
- Nickel inhalation increases the risk of cancer or of gastrointestinal symptoms.
- Exposure to inhalable nickel may result in chronic irritation of the upper respiratory tract or bronchial asthma.
- Nickel inhalation exposure increases susceptibility to respiratory infection, allergic contact dermatitis, and pulmonary edema.
- The main uses of nickel in industry include production of stainless steel, alloys, electroplating, coinage, and alkaline batteries.

top

LEAD

- Lead is known to cause cancer in animals.
- Lead may cause cancer in humans.
- Lead is toxic, and soluble in body fluids when inhaled.
- Lead interacts with enzymes, especially those associated with heme synthesis (blood).
- Absorption of low levels of lead causes an increase in blood pressure in humans.
- Lead causes anemia at blood levels above 80 ug/dl.
- Lead poisoning effects on the brain may not be reversible.

- Long term exposure to lead may lead to kidney disease.
- Lead is a possible Reproductive Toxin.
- Lead may affect sperm formation (at greater than 11.9 ug/dl blood lead).
- Lead exposure affects the development of fetuses. Children who were exposed to blood lead levels of greater than 10 ug/dl in the womb have been found to have developmental effects such as depressed intellectual development.
- Air to blood lead levels: 0.03 to 0.19 ug/dl blood per mg/m3 of lead in air.
- The main uses of lead in industry include alloys (solder, bronze, brass), paint pigments, storage batteries, glass, plastics, ceramics.

top

CADMIUM

- Cadmium is confirmed to cause cancer in humans.
- Cadmium primarily targets the kidneys.
- Chronic cadmium exposure is linked to gastrointestinal symptoms, anemia, rhinitis, discoloration of teeth, microfractures, pulmonary emphysema and kidney disease.
- The main industrial uses of cadmium include metal coatings, bearings, reactor control rods, storage batteries, television phosphors, semiconductors, pigments, and dry film lubricants.

top

CHROMIUM

- Cr VI compounds are recognized to cause cancer.
- Cr VI compounds can easily pass into the cell through the cell membrane.
- Cr VI compounds are sensitizers, and can therefore induce an allergic reaction in some individuals.

top

NITRIC OXIDE

- Nitric oxide reacts with haemoglobin to hinder oxygen uptake in the blood.
- Nitric oxide reacts with haemoglobin 1400 times more effectively than carbon monoxide reacts with haemoglobin.
- The toxicity of nitric oxide when combined with carbon monoxide (also in tobacco smoke) is additive.

top

PYRIDINE

- Pyridine vapour causes eye and upper respiratory tract irritation in humans.
- Exposure to pyridine results in an increased production of blood platelets.
- Longer duration exposure to pyridine causes nausea, headache, insomnia, nervousness, and abdominal discomfort in humans.
- The disagreeable odor of pyridine is detectable at less than 1 ppm.
- The main industrial uses of pyridine include solvents, pesticides and resins.

top

QUINOLINE

- Quinoline causes genetic mutations (mutagen) and therefore may increase your risk of cancer.
- Repeated exposure damages the retina of the eye, affecting vision.
- Repeated exposure to quinoline may damage the liver.
- Quinoline exposure may lead to allergy, with rash and itching (sensitizer).
- Quinoline is irritating to the eyes, nose, throat and bronchial tubes, and may cause sore throat, nose bleeds, cough and phlegm.
- Absorption occurs both by inhalation and through the skin.
- Quinoline bioaccumulates in the tissues of fish.
- The main industrial uses of quinoline include dyes, catalysts, insecticides, herbicides, corrosion inhibitors and to preserve anatomical specimens.

top

HYDROQUINONE

- Exposure to hydroquinone leads to eye injury, skin irritation and central nervous system effects in humans.
- The main uses of hydroquinone in industry include rubber production, photography, paints, varnishes and in motor fuel.

top

RESORCINOL

- Resorcinol was found to be irritating to skin and eyes in humans.
- The main industrial uses of resorcinol include tanning, photography, resins, dyes, laminates and adhesives.

top

CATECHOL

- Catechol, when inhaled with benzo[a]pyrene (also found in tobacco smoke), is co-carcinogenic.
- Catechol causes increased blood pressure, upper respiratory tract irritation and eczematous dermatitis in humans.
- At higher doses, catechol causes kidney damage and convulsions.
- The main uses of catechol in industry include photography, rubber, dye, oil, insecticides, and inks.

top

PHENOL

- Studies have shown phenol to be toxic to the respiratory, cardiovascular, hepatic, renal and neurological systems of animals.
- Higher doses of phenol may damage the lungs and central nervous system and induce convulsions in humans.
- Phenol is irritating to the skin, mucous membranes and eyes in humans.
- Phenol may be absorbed by inhalation or through the skin.
- The main industrial uses of phenol include chemicals and drugs, disinfectants, germicidal paints and slimicides.

top

CRESOL

- Cresol was found to promote tumors in mice.
- Cresol is strongly irritating to skin, and causes dermatitis in humans.
- Long term exposure to cresol leads to headaches, nausea, vomiting, elevated blood pressure, impaired kidney function, blood-calcium imbalance and marked tremors, in humans.
- Cresol is absorbed through the skin.
- The main uses of cresol in industry include ore flotation, disinfectants, synthetic resins, dyes, fumigants, and explosives.

top

TAR

Tar is the tobacco industry term for all non-gaseous, non-nicotine, non-water chemicals in tobacco smoke.

top

NICOTINE

- Free-base nicotine (in tobacco smoke) is absorbed almost instantly by inhalation, ingestion and skin contact.
- Nicotine concentrates in the brain, the kidney, the stomach mucosa, the adrenal medulla, the nasal mucosa and the salivary glands.
- Studies show that nicotine exposure can result in seizures, vomiting, depressions of the central nervous system, growth retardation, developmental toxicity in fetuses, and preterm birth with reduced body weight and brain development in animals.
- Nicotine is excreted in breast milk.
- Mild nicotine poisoning in humans results in the following symptoms: vomiting, diarrhea, increase in respiration, heart rate, blood pressure, headache, dizziness, and neurological stimulation.
- Nicotine is considered responsible for many of the acute psychological and physiological effects of smoking, chewing or inhaling tobacco.
- The main uses of nicotine in industry (besides tobacco) include insecticides (now mostly banned) and as tranquilizing darts for wildlife.

top

CARBON MONOXIDE

- Tobacco Smoke is the major source of personal inhalation of carbon monoxide.
- Carbon monoxide is absorbed into the blood, resulting in reduction in exercise tolerance, increased angina and headaches.
- Carbon monoxide binds to haemoglobin, reducing the oxygen-carrying capacity of the blood.
- As little as 3% absorbed carbon monoxide in haemoglobin results in decreased psychomotor function, and therefore can impair driving skills. Headaches may occur at 10% carbon monoxide in haemoglobin.
- Carbon monoxide binds to myoglobin, decreasing heart and muscle function.
- Carbon monoxide is a possible Reproductive Toxin.

- Studies on pregnant animals show decreased birth weights, fetal death or damage at moderate levels of carbon monoxide.
- Fetal carbon monoxide levels are generally 10 to 15% higher than maternal levels.
- Inhaled tobacco smoke increases the level of carbon monoxide in the fetus, increasing the chance of low birth weight, and possible perinatal death or retardation of mental abilities.

top

1, 3-BUTADIENE

- 1,3-butadiene suspected to cause cancer in humans.
- Joint exposure to styrene (also found in tobacco smoke) may increase the risk of disease.
- The toxicity of 1,3-butadiene is increased by prolonged or repeated exposures.
- The main industrial uses of 1,3-butadiene include synthetic rubber and tire manufacture.

top

ISOPRENE

- Isoprene causes skin, eye and mucous membrane irritation.

top

ACRYLONITRILE

- Acrylonitrile is suspected to cause cancer in humans.
- Acrylonitrile is highly toxic. It is similar to cyanide in toxicity, and is also known as 'vinyl cyanide'.
- Absorption of acrylonitrile is from the respiratory and gastrointestinal tract and through the skin.
- Studies on pregnant animals showed 'a significant maternal toxicity', leading to increased possibility of deformation in the fetus and offspring.
- The main industrial uses of acrylonitrile include manufacture of bottles and as a fumigant for tobacco.
- In the United States, acrylonitrile has been withdrawn as a fumigant for all other food commodities.

top

BENZENE

- Benzene is confirmed to cause cancer in humans.
- Benzene is known to cause leukemia in humans.
- Cumulative exposure to benzene is the most likely predictor of the possibility of developing leukemia.
- Leukemia may manifest 2 to 50 years after exposure to benzene.
- Benzene is highly toxic.
- Benzene produces chromosomal aberrations in humans and in animals.
- Benzene is absorbed through the skin.
- Previously, benzene was used in industry to manufacture inks, rubber, lacquers and paint remover.

top

TOLUENE

- Toluene is highly toxic.
- Toluene is a possible Reproductive Toxin.
- Inhaled toluene appears in blood circulation within 10 seconds and accumulates in body fat.
- Toluene is a depressant to the central nervous system in animals and in humans.
- Long term low level exposure results in headaches, lassitude, loss of appetite, disturbances in menstruation, reductions in intelligence and psychomotor skills.
- Higher exposure results in encephalopathy, headache, depression, lassitude, impaired coordination, transient memory loss, impaired reaction time, dizziness, nasal discharge, drowsiness, and metallic taste.
- The main uses of toluene in industry include rubbers, oils, resins, adhesives, inks, detergents, dyes, and explosives.

top

STYRENE

- Styrene is a possible human carcinogen.
- Styrene has been found to produce headaches, ocular and conjunctival irritation and slowed reaction time, fatigue, dizziness and nausea, reduced attention and manual dexterity, and reductions in colour discrimination, in humans.
- Reproductive effects of styrene include a possible increased incidence of spontaneous abortion and increased number of abnormal sperm.
- When styrene and butadiene (also in tobacco smoke) are combined, they produce 4-phylyglycolhexene, a suspected sensitizer.
- The main industrial uses of styrene include plastics,

coatings, polyesters, resins, and synthetic rubbers.

top

NNN

- NNN (N-nitrosornicotine) is a carcinogenic Tobacco-Specific Nitrosamine (TSNA) found only in tobacco products.
- NNN is formed from nicotine directly and is the most abundant cancer-causing TSNA.
- NNN is a yellow, oily liquid that is known to cause nose, throat, lung and digestive tract cancer in animals.
- NNN may cause reproductive damage in humans.-These is no safe level of exposure to NNN.

top

NNK

- NNK [(4-methylnitrosamino)-1-(3-pyridyl)-1-butanone] is a carcinogenic Tobacco-Specific Nitrosamine (TSNA) found only in tobacco products.
- NNK is a powerful lung carcinogen.
- NNK induces adenoma and AC tumors of the lung.
- There is no safe level of exposure to NAT.

top

NAT

- NAT (N-nitrosoanatabine) is a possibly carcinogenic Tobacco-Specific Nitrosamine (TSNA) found only in tobacco products.

top

Physicians for a Smoke-Free Canada
1226A Wellington Street
Ottawa, Ontario, Canada
K1Y 3A1
613-233-4878

You Are Stronger Than Nicotine

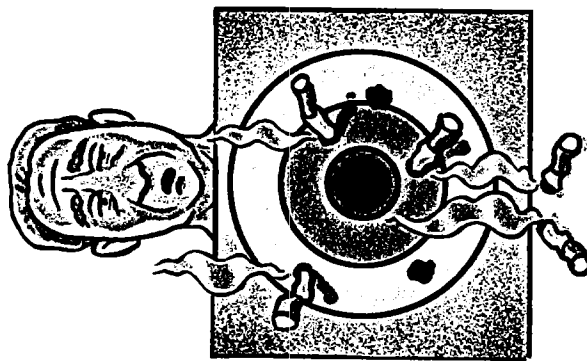
You have a drug addiction. Your drug is nicotine. Though legal, it is just as addictive and harmful as heroin or cocaine. The tobacco industry has made a fortune keeping you hooked. Will tobacco keep controlling you, or will you control it? The choice is yours. Over a million smokers a year quit. So can you. This booklet will show you how.

Your Old Friend Has Betrayed You

Your cigarette has always been a comfort. It has been with you every step of the way for years. But those days are over. Smoking has turned out to be your enemy. It has already made you sick. Unless you quit, it will only make you sicker. And it may well kill you.

Will You Dodge the Bullet Next Time?

You came through this health crisis. Next time you might not. Quitting will mean replacing a lot of old habits with new ones. That will be hard. But not as hard as getting sicker and sicker. You can stop poisoning yourself. You must. Not tomorrow. Not next week. Today.



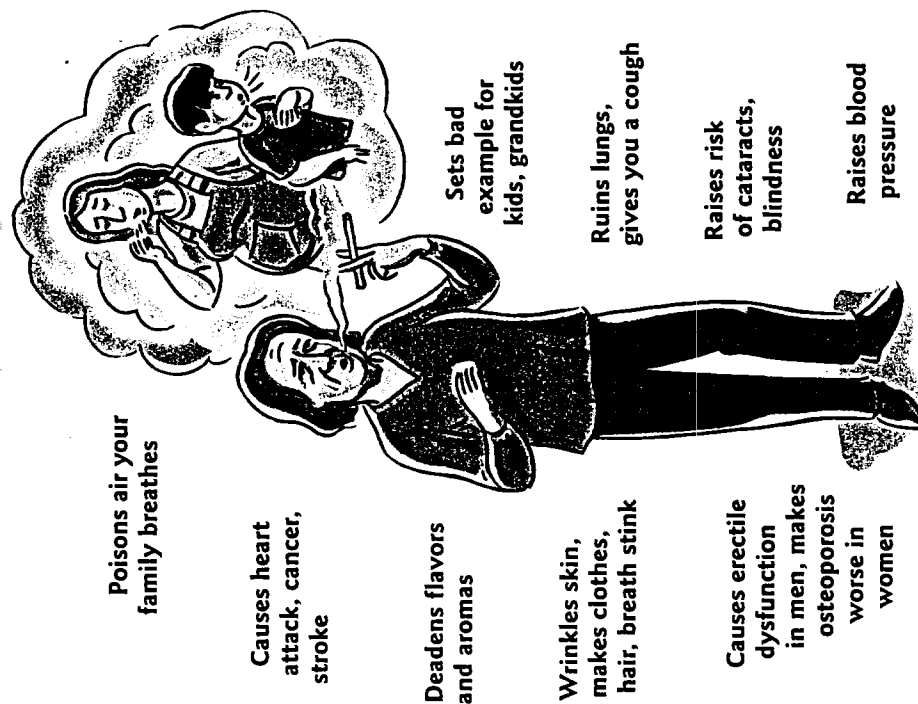
Smoking targets you for heart disease.

This booklet is not intended as a substitute for professional medical care. Only your doctor can diagnose and treat a medical problem.

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What Smoking Does to You

Smoking is slow suicide. It poisons your body. It makes you unattractive. And it robs you of many of life's pleasures. Would you stick with any other activity that did all that?



How Quitting Helps You

As soon as you quit, your body starts to heal. Breathing gets easier. Your stamina and your senses of taste and smell start coming back. Quitting lowers your odds of getting or dying of smoking-related diseases. And it saves your loved ones from breathing your smoke.

Cervical cancer tied to secondhand smoke

by Katrina Woznicki
Womens e-News

NEW YORK (IPS/GIN)— Women exposed to secondhand smoke increase their risk of developing cervical cancer, according to a just-published study from Johns Hopkins Bloomberg School of Public Health.

The study published in the January issue of *Obstetrics and Gynecology* could have critical health implications, as public health advocates work to not only educate women about reducing their risks for cervical cancer, but also lower tobacco use around the globe.

"The evidence is strong," said lead researcher Anthony Alberg, an assistant professor in the Department of Epidemiology. "The findings should encourage smokers to quit and warn non-smokers who live with smokers to decrease their secondhand smoke exposure."

While researchers have long suspected that secondhand smoke raised cervical cancer rates, the study—one of the biggest in the United States—makes the link more definitive. The results are being seen as especially important for women living in developing countries, where smoking is on the

rise and cervical cancer is a leading cause of death.

Prof. Alberg and his team examined the exposures of 51,173 women age 25 and older in Washington County, Md., to household smoking in 1963 and then 1975. The women filled out questionnaires about their exposure to cigarette smoke, who currently or formerly smoked in their households, household member ages, years of education and marital status. Each group was followed 15 years. Researchers then compared women who lived with nonsmokers to women who lived with smokers and monitored who developed cervical cancer. Investigators found that women exposed to passive smoking faced a 2.1-fold increased risk for the disease in 1963. By the 1975 study group, the figure dropped to a 1.4-fold greater risk.

Prof. Alberg said he was "puzzled" by this drop and had "no clear explanation for it." He speculated that one possible reason was that the women in the 1975 group were working outside of the home and may have reduced their household exposure to tobacco. William Au, professor in the Department of Preventive Medicine and Community Health at the University of Texas Medical Branch in Galveston, Texas, said the study,

which was peer-reviewed, proves a conclusive link.

"This is a well-conducted study based on scientific protocol and it has tremendous implications to human health," he said. "We're now seeing how low levels of toxic substances such as secondhand smoke can cause cancer in the human population."

Passive smoking has been known to increase the risks for heart disease and lung cancer in both men and women, and active cigarette smoking has been long established as a major risk factor for cervical cancer. Although scientists have suspected a link between secondhand smoke and cervical cancer, they needed more data to prove it.

One of the more recent studies came from Singapore and was published in the April issue of *Gynecologic Oncology*. Researchers studied 623 women and found their risk of certain abnormal cervical cells that signal the possible onset of cervical cancer increased by 4.6 percent for every cigarette the woman's spouse smoked.

"It's really important people get the message that smoking does much more than we ever thought that it did and that it affects our health in ways we don't even fully know about yet," said Hollis Forster, executive director

of the National Cervical Cancer Coalition, a nonprofit organization in Berkeley, Calif.

Special risks for minorities

Twenty-two percent of the U.S. population smoked in 2003, down from 24 percent in 1998, according to the federal Centers for Disease Control and Prevention (CDC). The American Cancer Society reports that there are more than 10,000 cases of invasive cervical cancer every year in the U.S. and the disease claims 3,900 lives. Black women are most vulnerable to cervical cancer, according to the CDC.

The CDC reports from 1992-2000, only 62.6 percent of Black women survived cervical cancer five years after being diagnosed, compared to a survival rate of 73.3 percent among White women. The federal agency also reports higher Pap smear testing, the gold standard of screening for cervical cancer, among White women.

While smoking rates have been dropping steadily in this country and cervical cancer rates have followed suit thanks to detection with Pap smear tests, the rates for both remain a serious public health threat elsewhere around the world.

In developing nations, cervical cancer is the second-leading cause of cancer deaths among women, after lung cancer with 80 percent of

the 500,000 new cases every year occurring in Latin America, Africa, and Southeast Asia.

Although easily treated if detected, cervical cancer remains a top public health threat because of human papillomavirus (HPV), a sexually transmitted infection that causes the disease. Prof. Alberg said he suspects tobacco exposure may exacerbate HPV infection. "It is possible that cigarette smoke acts in concert with HPV to promote progression to cancer," he said.

Women, however, should not think being around cigarette smoke will directly result in HPV infection. While smoking is unrelated to the acquisition of HPV infection, it is "related to immunity, which is important in the progression" of cervical tumors, said Janet Daling, an investigator at the Fred Hutchinson Cancer Research Center in Seattle.

The rising number of public smoking bans may make it easier for women.

"The banning of smoking in public places is just the beginning," said Prof. Au. "First, it's the ban. Second, it's education. We don't want people to quit smoking in public places and then just smoke in the home. That puts family members and children at risk."

USA TODAY • WEDNESDAY, MARCH 9, 2005 • 7

Firestorm could be brewing over secondhand smoke

Breast cancer connection is complicated

By John Ritter
USA TODAY

SAN FRANCISCO — Cancer scientists are split over whether smoking causes breast cancer, but they agree on one thing: The debate is far more complex than linking smoking to lung cancer or heart disease.

The U.S. surgeon general says tobacco smoke — whether secondhand or inhaled by smokers — can cause both those killers. Only the tobacco industry disputes the evidence. But breast cancer, a disease that strikes 270,000 U.S. women a year, is another matter. Though a California government report is the first to affirm secondhand smoke as a cause, it's far from the last word.

Chemicals in cigarette smoke cause breast cancer in rats; the chemicals are found in human breast tissue. Recent studies of groups of women show a breast cancer-smoking link. But science has been slow — too slow, breast cancer advocates say — to indict tobacco.

"If we spend this much time looking at each chemical out there that could cause breast cancer or other cancers, we'll all be dead before the analysis is completed," says Nancy Evans, a health science consultant with the Breast Cancer Fund, a national group that focuses on prevention.

Scientific caution is partly a result of Big Tobacco's clout. "The tobacco industry is so wealthy and powerful that you want what you say to be incontrovertible," says Michael Thun, who heads the American Cancer Society's epidemiological research.

The industry disputed the California findings in public comments included in the report. Three tobacco companies declined interview requests.



Taking action since 1966

California's Air Resources Board has been a national leader in curtailing pollution, particularly from vehicles:

► **1966:** A pollution-control board that was the ARB's forerunner was the first agency in the nation to set tailpipe-emissions standards for cars. By the time the federal Clean Air Act passed, creating the Environmental Protection Agency four years later, more than 1 million California cars had early pollution-control devices.

► **1970:** The ARB required automakers to meet the first standards to control smog-forming hydrocarbon and nitrogen oxide emissions.

► **1976:** With invention of the catalytic converter, a technology to cut tailpipe emissions, the ARB made California the first state to phase out leaded gasoline, a requirement for converters to work.

► **1978:** Cleaner gasoline allowed the ARB to toughen emissions standards to the point that automakers had to equip cars with catalytic converters, first for cars sold in California, later nationwide.

► **1990:** The ARB made California the first state to require an oxygen additive in gasoline to cut carbon monoxide emissions. Within a year after the fuel became available, CO emissions had dropped by 10% statewide.

But a bigger reason is uncertainty about the data. California scientists who concluded that secondhand smoke causes breast cancer and whose report is likely to be approved next week by a review panel were persuaded by "the weight of evidence."

Much of that was newer, better studies, says Melanie Marty, the section chief with the Office of Environmental Health Hazard Assessment who supervised the report. "What you want is multiple studies that show an effect," she says. "As time has gone by, more and more have shown an effect."

Marty's team looked at older studies that "didn't ask enough questions to figure out who was really exposed (to secondhand smoke) and who wasn't." But in six recent studies that were careful to take women who'd been exposed out of control groups, the risks went up, she says.

The scientists also saw a breast cancer link to active smoking in the newer studies, though not as distinct as with secondhand smoke. That was important, Marty says, because the scientific consensus has been that active smoking doesn't put women at risk. The California scientists didn't calculate the risk for active smoking.

The lower risk seen for active smoking, which bathes tissue with more carcinogens than secondhand smoke, is probably because of estrogen, Marty says. The female hormone raises breast cancer risk, but a leading theory is that big doses of inhaled smoke blunt its ability to fuel tumor growth, while smaller secondhand doses don't.

Health risks to children

Women exposed to secondhand smoke have a 26% to 90% higher risk of breast cancer, the report says. That broad range is due to wide disparity in exposure — a woman married to a three-pack-a-day smoker for 30 years vs. a woman exposed for a short time. The greater the exposure, the earlier the age of exposure — particularly before puberty and a first pregnancy — the higher the risks, the report said.

The California scientists gave more weight to toxicology — whether chemicals in smoke cause breast cancer in



By Chris Purlong, Getty Images

The ban bandwagon

States that enacted smoking bans for workplaces, bars or restaurants, or for all three:

- | | |
|----------------|-----------------|
| ► California | ► Connecticut |
| ► Utah | ► Maine |
| ► South Dakota | ► Idaho |
| ► Delaware | ► Massachusetts |
| ► Florida | ► Rhode Island |
| ► New York | ► Vermont |

Source: USA TODAY research; American Nonsmokers' Rights Foundation

lab animals — than the surgeon general or the International Agency for Research on Cancer. Toxicology provides "biological plausibility," Marty says. "If studies don't bring it forward as a reason why all these things make sense, they're missing a piece of the puzzle."

Whether the California breast cancer findings — and newer studies they're partly based on — influence a surgeon general's report on secondhand smoke due this year is uncertain.

"I'd be very surprised to see that

change," says Barbara Brenner, executive director of Breast Cancer Action. "There's more caution in the scientific community than is necessary in the interest of the public's health. What science understands as proof is almost an ever-retreating goal."

The National Cancer Institute published the Air Resources Board's widely praised 1997 secondhand smoke report. It found evidence of a breast cancer link inconclusive. "We need to take this new report seriously, look at it closely," says Deborah Winn, chief of an NCI epidemiology branch.

Even if the review panel approves the new report, the board may not. It took no action and forwarded the 1997 report to the state health department, deciding it had no authority to regulate indoor pollution.

But the new report has measurements on outdoor secondhand smoke from several California locations. An amusement park had the highest nicotine concentrations. Lawyers are researching whether the board can ban smoking in vehicles carrying children,

spokesman Jerry Martin says.

A bill to do that failed narrowly last year in the California Legislature after heavy tobacco industry lobbying.

The board might find a rationale now. In 1999, the Legislature expanded its scope, ordering it to assess pollutants' health risks to children because of their greater susceptibility. Other than private homes and a few workplace exceptions, vehicles are the only major category of enclosed space where smoking is permitted in California.

"It's fair to say there's some interest in going further than they did in 1997," Martin says. The 1967 law that created the board says it must act to protect public health even without "undisputed scientific evidence."

No states prohibit smoking in vehicles. "That would be significant," says Brenner of Breast Cancer Action. "The more we restrain where people smoke publicly, the more likely they are to smoke in the places where they can — homes and cars."

► Study finds breast cancer link, 1A

► **1994:** The ARB required the advanced diagnostic sensing systems found on all new cars today, including sensors to detect emissions from a vehicle's air conditioning.

► **1998:** The ARB classified increasingly large SUVs, minivans and light pickups as autos to prevent automakers from taking advantage of the state's more lenient truck-emissions standards.

► **2004:** A state law permitted the ARB to regulate the nation's first regulations requiring automakers to cut greenhouse-gas emissions from vehicles, by 22% in 2012 and by 30% in 2016.

Reporting by John Ritter

USA TODAY Snapshots

Top cigarette producers globally

Countries that produced the most cigarettes in 2003 (in billions of cigarettes): China 1,735



By Ashley Barnard and Marry E. Mullins, USA TODAY

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Secondhand smoke causes breast cancer, study says

Calif. finding could have wide effect

By John Rimer
USA TODAY

SAN FRANCISCO — Scientists at an influential California agency have concluded that secondhand smoke causes breast cancer, a finding that could have broad impact on cancer research and lead to even tougher anti-smoking regulations.

Although recent studies have linked smoking to breast cancer, no major public health group, including the American Cancer Society, the Centers for Disease Control and Prevention and the National Cancer Institute, has declared it a cause of the disease that kills 40,000 women each year in the USA.

The finding by scientists for the Air Resources Board — whose early efforts to regulate auto emissions were a model for the rest of the country — could fuel workplace smoking bans in more states. And it is likely to refocus the scientific debate over the link between smoking and breast cancer.

"I have to say without reservation it will stimulate continued and accelerated scientific evaluation of the smoking and breast cancer issue," says Terry Pechacek, associate director for science in the CDC's office on smoking and health.

A scientific review panel is expected to approve the report as early as Monday and forward it to the Air Resources Board, which has broad state authority to regulate air pollution.

The 1,200-page report analyzes new data on the extent of Californians' exposure to secondhand smoke and more than 1,000 studies of health effects from secondhand smoke.

The conclusion that secondhand smoke causes breast cancer, particularly in younger women, challenges conventional scientific thinking because most studies, until recently, had found no connection between female smokers and breast cancer.

But California scientists based their conclusion on recent human studies that they determined had more careful assessments of long-term exposure to tobacco smoke. The report also gave more weight to toxicology evidence from animal studies than previous studies by the surgeon general and others. It's well-documented that chemicals from cigarettes cause breast cancer in lab animals.

Cancer risk

A woman's risk of developing breast cancer increases with age:

30 to 40: 1 in 227
40 to 50: 1 in 67
50 to 60: 1 in 36
60 to 70: 1 in 26

Source: National Cancer Institute

Breast cancer link

■ Debate not over, 7D

Overall, women exposed to secondhand smoke have up to a 90% greater risk of breast cancer, the report says. It says secondhand smoke kills as many as 73,400 a year in the USA.

The report did not estimate

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The report did not estimate

"It's controversial," Samet says. "Concluding that passive smoke causes breast cancer has potentially powerful implications for tobacco control and breast cancer control. So there has been tension over it."

The report did not estimate

African-American Male Smokers Are More at Risk of Getting Cancer

By WILLIAM M. BULKELEY

African-American male smokers are much more likely to get lung cancer than are white or Latino men who smoke, researchers reported in a study in the New England Journal of Medicine to be published today.

The study, which found that native Hawaiian smokers had high lung-cancer rates while Japanese Americans had low rates, didn't attempt to find reasons for the differences. An accompanying editorial said it isn't clear the information has public health value, since "eliminating smoking would largely reduce and equalize the rates of lung cancer" across all groups.

Nevertheless, according to the editorial, the study emphasized that ethnic information may be useful in diagnosing or treating some illnesses. Doctors and researchers have become increasingly aware that many diseases have different impacts in different racial and ethnic groups. Last year, the Food and Drug Administration, for the first time, approved a drug designed for use by one racial group when it approved NitroMed Inc.'s BiDil to combat heart disease in African-Americans.

African-American groups, including Congress's Black Caucus and various physicians associations, have been pressing for more studies of racial differences in disease incidence and treatment as well as whether drug effectiveness differs by racial groups.

The lung-cancer study was conducted by researchers at the University of Southern California and the University of Hawaii, using data from the Multiethnic Cohort Study, a database of 215,000 individuals who volunteered their ethnic identities for medical research purposes. For the cancer study, researchers followed 183,000 people in an eight-year period starting in 1993. Of the total, 1,979 got lung cancer.

According to the study, African-American men who smoked had 264 cases of lung-cancer per 100,000 individuals, compared to 158 cases in white men and 79 cases in Latino men. Native Hawaiian men had 264 cases and

Japanese American men had 121 cases. Other ethnic groups weren't included in the study. Women had lower incidences of lung cancer, but ethnic differences generally followed the same pattern. The differences were most pronounced among people smoking 20 or fewer cigarettes a day. The researchers said ethnic and racial differences largely disappeared among heavy smokers who had 30 or more cigarettes a day.

Christopher Haiman, an assistant professor in the Keck School of Medicine at the University of Southern California, who led the study, said the effects he found were more pronounced than smaller previous studies. He said "the next step is to understand more about the mechanism to try to explain the differences." He said both genetic

Ethnic and racial differences largely disappeared among heavy smokers.

and behavior differences could affect the way smoking causes lung cancer. For example, he said, there could be group differences in "smoking behavior such as depth or frequency of inhalation." There also could be genetic differences that affect the way people metabolize the smoke.

Neil Risch, director of the Institute for Human Genetics at University of California at San Francisco, who wrote the editorial accompanying the study, said the lung cancer results "provide an example of how ethnicity can interact with environmental factors in terms of the risk of disease." He said other studies have found that Asians and Latinos have lower rates of most cancer except for stomach cancer. He added that other diseases are likely to have similar variations. "There's probably a mix of genetic and environmental factors," he said.

SOCIAL SCIENCE

WSS 1/19/07

Behind Black Smokers' Preference for Menthol

[RADAR ONLINE • JAN. 15]

Big Tobacco has played a part in drawing one of the more subtle racial lines in America: Three out of four African-American smokers smoke menthol cigarettes, while only one out of four white American smokers do.

Sarah S. Lochlann Jain, an assistant professor of cultural anthropology at Stanford University, tells Radar Online that the disparity might have originated in the 1920s, when accessible eucalyptus- or menthol-laced cold medications were popular with African-Americans.

But the real gap opened in the 1960s after Big Tobacco hatched a successful plan to build on menthols' popularity with a long-term marketing strategy. Tobacco companies placed ads for menthols in magazines like Ebony and Jade.

The differences in preferences might have health ramifications, Radar Online says research has suggested that the anesthetic sensation from menthols could encourage smokers to inhale more deeply, increasing the deadliness. This may compound African-Americans' greater susceptibility to lung cancer from smoking.

WED, 12/21/05

WSJ - Sec. D

Using X-Rays to Find Lung Cancer

By JENNIFER CORBETT DOOREN

Dow Jones Newswires

WASHINGTON—Screening for lung cancer with chest X-rays can detect lung cancer at an early stage, but it is too soon to say whether the tool would prevent lung-cancer deaths, according to results of a government-funded study looking at cancer-screening methods.

Currently, most lung cancer is diagnosed in advanced stages and most patients die within two years of being diagnosed. There is no routine method at present for detecting lung cancer, the leading cancer killer of both men and women, at an early stage, unlike cancers of the breast, prostate and colon.

Researchers, led by the National Cancer Institute, part of the National Institutes of Health, wanted to know if routine chest X-rays could pick up the lung cancer sooner. The study will be published in today's edition of the *Journal of the National Cancer Institute*.

From 1993 to 2001, researchers enrolled 154,942 men and women who were aged 55 to 74, including current smokers, former smokers and those who had never smoked.

The participants are enrolled as part of a larger prostate, lung, colorectal and ovarian cancer screening trial that is looking at cancer-screening methods. About 67,000 men and women in the lung-cancer portion of the study received an initial chest X-ray.

The results showed that X-rays detected 126 cases of lung cancer, almost half of which were at stage one, considered the earliest stage.

However, Christine Berg, who oversees the PLCO trial, said the study showed the chest X-rays produced a high false-positive rate. The X-rays detected spots or tissues on the lungs of almost 6,000 patients requiring them to undergo additional testing. The 126 cases of lung cancer were confirmed after additional testing.

WSS 8/16/05

Even Among People Who Never Smoked, Diagnoses of Lung Cancer Are Rising

LUNG CANCER HAS always been viewed as a disease of smokers; or even ex-smokers. But now, doctors say the deadly cancer may be showing up more often in patients who never smoked at all.

The disease is still a far bigger worry for smokers and former smokers, but there's good reason to take a closer look at so-called never-smokers with lung cancer. Recently, researchers discovered that female never-smokers are far more likely to respond to the new targeted therapies for lung cancer — Iressa and Tarceva.

Understanding the genetic differences between lung cancers in never-smokers and those who have smoked could ultimately lead to better treatments for both groups, notes Bruce E. Johnson, director of thoracic oncology at Dana-Farber Cancer Institute. Dana-Farber is conducting a study of the targeted drug Tarceva as a first-line treatment in female lung-cancer patients who never smoked.

The fact that never-smokers are also at risk for lung cancer was highlighted last week when Dana Reeve, the widow of actor Christopher Reeve, announced she was undergoing treatment for lung cancer. Ms. Reeve never smoked.

The hard data on never-smokers are sketchy. But doctors who treat lung-cancer patients say they believe the number of never-smokers with the disease is growing. "There's no question in my clinic, week to week, month to month, there are more never-smokers," says Scott J. Swanson, chief

of thoracic surgery at Mount Sinai School of Medicine in New York.

Right now, doctors say about 15% of patients diagnosed with lung cancer each year fall into the category of never-smokers—that amounts to about 28,500 people.

Nobody really knows why lung cancer appears to be increasing in never-smokers. One theory is that baby boomers may have been exposed to large amounts of second-hand smoke by parents and grandparents who smoked at home. But doctors may not think of lung-cancer risk when treating patients who never smoked, and may attribute symptoms like chronic cough to other causes. Lung-cancer experts say doctors and nurses need to be more vigilant in taking a patient's personal smoke-related history, including whether they come from families with smokers.

Many cases of lung cancer in never-smokers are detected by accident. That was the case for Cynthia Kneibert, a 65-year-old psychotherapist from Sedalia, Mo., who underwent a routine chest X-ray to prepare for hip-replacement surgery. After finding a mass in her lung, doctors removed part of her lung and Ms. Kneibert is now undergoing chemotherapy.

Ms. Kneibert, who exercised regularly and considered herself the picture of health, was stunned that she had lung cancer, sending her and her husband on a search for what might have caused it. Except for a few experimental puffs as a teenager, Ms. Kneibert never smoked. But her father did. "I remember riding in cars as a little child with the windows up and the car filled with smoke," she says.

Other Factors

Some risks for lung cancer in people who never smoked:

RISK	EXPOSURE
Second-hand smoke	30% higher risk in nonsmokers married to smokers
Radon	Residential exposure increases risk between 11% and 21%
Workplace	Asbestos, diesel exhaust and other chemicals pose risk
Family history	Chromosomal abnormality increases risk
Being female	Hormonal difference may put women at higher risk

Source: American Cancer Society

Also, Ms. Kneibert lives in an area known to have high levels of radon. Radon is a naturally occurring radioactive, invisible, odorless gas. It can accumulate in enclosed areas, such as homes and underground mines, and it's known to increase risk for lung cancer.

Ms. Kneibert learned her basement had a level of seven picocuries of radon per liter of air. Research has shown that lung-cancer risk increases at residential concentrations of about three picocuries. But Ms. Kneibert spent little time in her basement, so it isn't clear whether she really had much radon exposure.

Most never-smoking patients will never know for sure whether the cancer was caused by a combination of passive smoke, radon or some other environmental exposure—or whether, for them, lung cancer was genetically determined, says Derek Raghavan, chairman of Cleveland Clinic's Taussig Cancer Center. Family history can put

you at higher risk, but scientists are also discovering that some people carry certain genetic mutations that may play a role.

Doctors do know that lung cancers in patients who never smoked are genetically different from those in smokers and former smokers. Never-smokers' tumors are more likely to carry the genetic signature that targeted therapies like Iressa are designed to attack.

Women never-smokers may also be at higher risk than men never-smokers. Part of the reason may simply be that there is a greater pool of women who have never smoked than men. But recent research has also shown that the mechanism by which the body repairs cell damage doesn't seem to work as well in women as in men, perhaps making them more vulnerable to cell damage from environmental factors. In addition, researchers are studying whether hormonal factors play a role in lung cancer.

Some studies support the idea that using special CT scans to screen smokers and former smokers can lower the death rate from lung cancer, but even less is known about how or whether to screen people who have never smoked. "Perhaps we should do a one-time screening of nonsmoking women at the age of 40—that's a question we need to study," says Claudia Henschke, radiology professor at New York-Presbyterian/Weill Cornell Medical Center.

Patients, regardless of smoking history, should always talk to a doctor about a persistent or unusual cough. And make sure your doctor knows if you have a family history of lung cancer, or you have been exposed to high levels of passive smoke, asbestos or other environmental risks. Finally, check out www.epa.gov/radon to learn more about radon and whether your home should be tested.

Email me at healthjournal@wssj.com; read my responses in Health Mailbox inside this section.

WEDNESDAY, FEBRUARY 1, 2006 **D5**

Heart Disease Evades Detection In Many Women Despite Signs

Associated Press

WASHINGTON—Conventional tests won't uncover heart disease in as many as three million U.S. women—because instead of the usual, bulky clogs in main arteries, these women have hard-to-spot buildups in smaller blood vessels, researchers said. These are the women who come to the doctor complaining of chest pain or shortness of breath but sometimes are sent away undiagnosed, not knowing they are actually at high risk for heart attacks in the next few years.

"The No. 1 message for women is, 'Pay attention to your symptoms,'" said Dr. George Sopko, a heart specialist at the National Institutes of Health, which sponsored the research. "If you don't have visible blockages, that doesn't mean you're not at risk."

Heart disease is the nation's leading killer of both men and women. In fact, slightly more women than men die from cardiovascular diseases each year—more than 480,000 of them, according to the American Heart Association.

Scientists are struggling to understand gender disparities: Women are less likely to receive aggressive treatment for heart

disease than men, are less likely to survive heart surgery and respond differently to different risk factors and therapies. They frequently have different heart-attack symptoms than men, such as fatigue instead of chest pain radiating down the arm.

Even the test considered best at diagnosing heart disease—angiography, which lets doctors watch as blood flows through key arteries—is less accurate for women than for men.

Reviewing clues from some recent research, the NIH's National Heart, Lung and Blood Institute highlighted why many women are at risk after a misleadingly "clear" angiogram. In a study called the Women's Ischemia Syndrome Evaluation, researchers have found that about two-thirds of women with chest pain pass an angiogram. But about half of them turn out to have a condition named "coronary microvascular syndrome," where plaque evenly coats very small arteries instead of forming more obvious obstructions in larger ones.

Angiograms can't see these tiny arteries, Dr. Sopko explained. The narrowed small arteries mean less oxygen flow to the heart, explaining the women's chest pain.

Attention Deficit May Be Tied to Smoking, Lead

Associated Press

CHICAGO—About one-third of attention-deficit cases among U.S. children may be linked with tobacco smoke before birth or to lead exposure afterward, according to provocative new research.

Even levels of lead the government considers acceptable appeared to increase a child's risk of having attention-deficit hyperactivity disorder, the study found.

It builds on previous research linking attention problems, including ADHD, with childhood lead exposure and smoking during pregnancy, and offers one of the first estimates for how much those environmental factors might contribute.

"It's a landmark paper that quantifies the number of cases of ADHD that can be attributed to very important environmental exposures," said Dr. Leo Trasande, assistant director of the Center for Children's Health and the Environment at Mount Si-

nai School of Medicine in New York.

The study bolsters suspicions that low-level lead exposure "is in fact associated with ADHD," said Dr. Trasande, who wasn't involved in the research.

"The findings of this study underscore the profound behavioral health impact of these prevalent exposures," said the authors, led by researcher Joe Braun of the University of Wisconsin-Milwaukee. The study was to be published online today in the journal *Environmental Health Perspectives*.

ADHD is a brain disorder affecting between 4% and 12% of school-age children—or as many as 3.8 million youngsters. Affected children often have trouble paying attention, and act impulsively.

Dr. Helen Binns, a researcher at Children's Memorial Hospital in Chicago, said the study is thoughtful but doesn't prove lead exposure is among the causes.

It is possible, for example, that children with ADHD are more likely than other to eat leaded paint chips or inhale leaded paint dust because of their hyperactivity.

The researchers analyzed data on nearly 4,000 U.S. children ages 4 to 11 who were part of a 1999-2002 government health survey. Included were 135 children treated for ADHD.

Children whose mothers smoked during pregnancy were 12 and a half times more likely to have ADHD than children who weren't prenatally exposed to tobacco.

Children with blood lead levels of more than two micrograms per deciliter were four times more likely to have ADHD than children with levels below 0.5 microgram per deciliter. The government's "acceptable" blood lead level is 10 micrograms per deciliter, and an estimated 310,000 U.S. children ages one to five have levels exceeding that.

Health Journal / By Tara Parker-Pope

Death of Ann Richards Stirs Debate About Esophageal-Cancer Screening

FORMER TEXAS GOV. Ann Richards's brief battle with esophageal cancer has raised new questions about whether more people should be screened for the deadly disease.

Esophageal cancer is one of the fastest-rising forms of cancer, but it is rarely detected early, when it is most curable. Most patients live less than a year after being diagnosed—just 15% survive for at least five years. Gov. Richards lived for just six months after her diagnosis.

The form of cancer that killed Gov. Richards—squamous cell—often is associated with a history of heavy alcohol and tobacco use. Gov. Richards was a recovering alcoholic and former smoker. The fastest-rising form of esophageal cancer—adenocarcinoma—is strongly linked with years of chronic heartburn and acid-reflux disease.

But whether people should be screened for the disease remains a matter of heated debate in the medical community because it isn't clear whether screening would be cost effective or make a meaningful difference in long-term survival rates. The medical community routinely screens for a range of diseases in high-risk populations, but there's no routine screening for people at high risk for "one of the deadliest tumors known—esophageal cancer," says Scott Swanson, chief of thoracic surgery at Mount Sinai School of Medicine in New York. "We need to consider [screening] for high-risk groups."

The best way to screen for esophageal cancer is with an endoscopy. During an endoscopy, the patient is sedated while a lighted tube is put down the throat to look for changes in the esophagus that might signal cancer. The procedure costs about \$1,000 and is typically covered by insurance. Although an endoscopy is considered a safe procedure, there are always risks associated with anesthesia and a slight risk of bleeding or infection after the procedure. The main concern about screening endoscopy is cost and that fact that the vast



Former Texas Gov. Ann Richards lived for just six months after her esophageal-cancer diagnosis.

majority of screening endoscopies wouldn't find cancer, but would tax the resources of an already overburdened health-care system.

Currently there are more than 14,000 cases of esophageal cancer each year. Considering that there are 40 million current smokers, 100 million former smokers and an estimated 40 million people who suffer weekly bouts of heartburn, the question is who among these groups is at highest risk for esophageal cancer and should be screened.

There are no simple answers. But we do know risk for the squamous-cell esophageal cancer is dramatically higher with long-term use of both tobacco and alcohol. It's six times more likely to occur in African-American men than in whites.

We also know that people with chronic heartburn—or gastroesophageal reflux disease, known as GERD—are at higher risk for esophageal cancer. Many doctors believe patients with a history of chronic heartburn for five years or more should be screened for precancerous changes in the lining of the esophagus—a condition known as Barrett's esophagus. It has been estimated that from 5% to 10% of Barrett's patients will go on to develop cancer. Eating a diet low in fruits and vegetables, or weighing 20 to three pounds more than your ideal

weight, also has been linked with increased risk for esophageal cancer.

Most doctors agree that patients already diagnosed with Barrett's should get regular screenings for cancer. The more difficult question is who among GERD sufferers should undergo screening endoscopy. Some researchers have suggested a one-time screening for anyone who has chronic reflux, particularly if the problem is serious enough to warrant long-term drug therapy. Others believe only those at highest risk should be screened—men older than 40 who have suffered reflux symptoms at least twice a week for five years. Patients who develop GERD for the first time after age 65 also should consider screening.

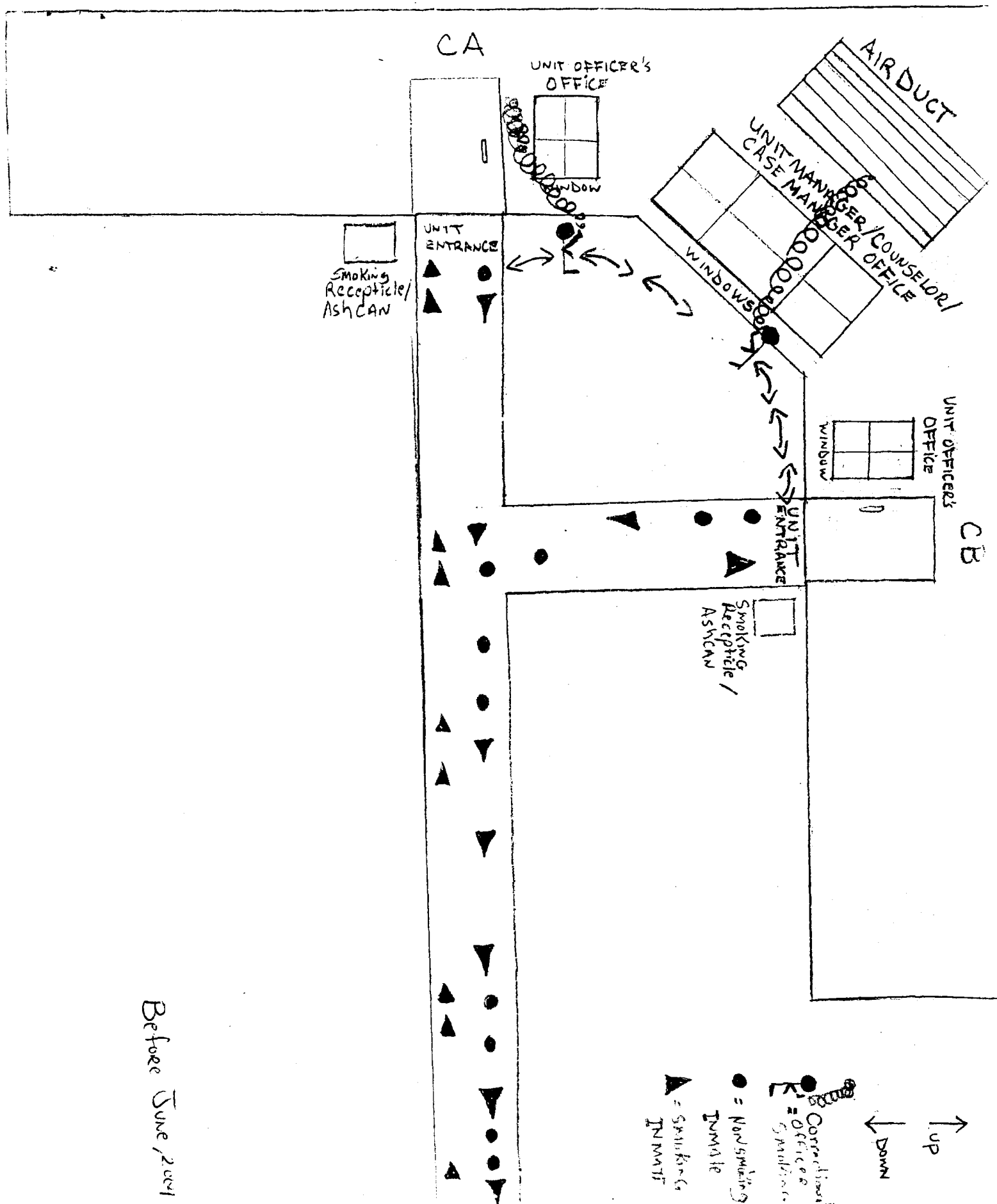
One answer to the screening question is to perform a screening endoscopy at the same time a patient is undergoing a screening colonoscopy, which typically happens around the age of 50. The patient already is sedated and the gastroenterologist who performs the colonoscopy also typically specializes in endoscopy. Some studies have shown that Barrett's is detected in as many as one out of four patients screened this way.

Finding esophageal cancer early makes a dramatic difference in survival because patients can qualify for surgical removal of the esophagus and sometimes the surrounding lymph nodes. An April study published in the *Journal of the American College of Surgeons* reviewed the medical records of 263 patients who underwent the surgery from 1992 through 2002. The overall survival rate during the last five years of the study was 50.4%, and jumped to 81% for patients who were diagnosed at Stage I.

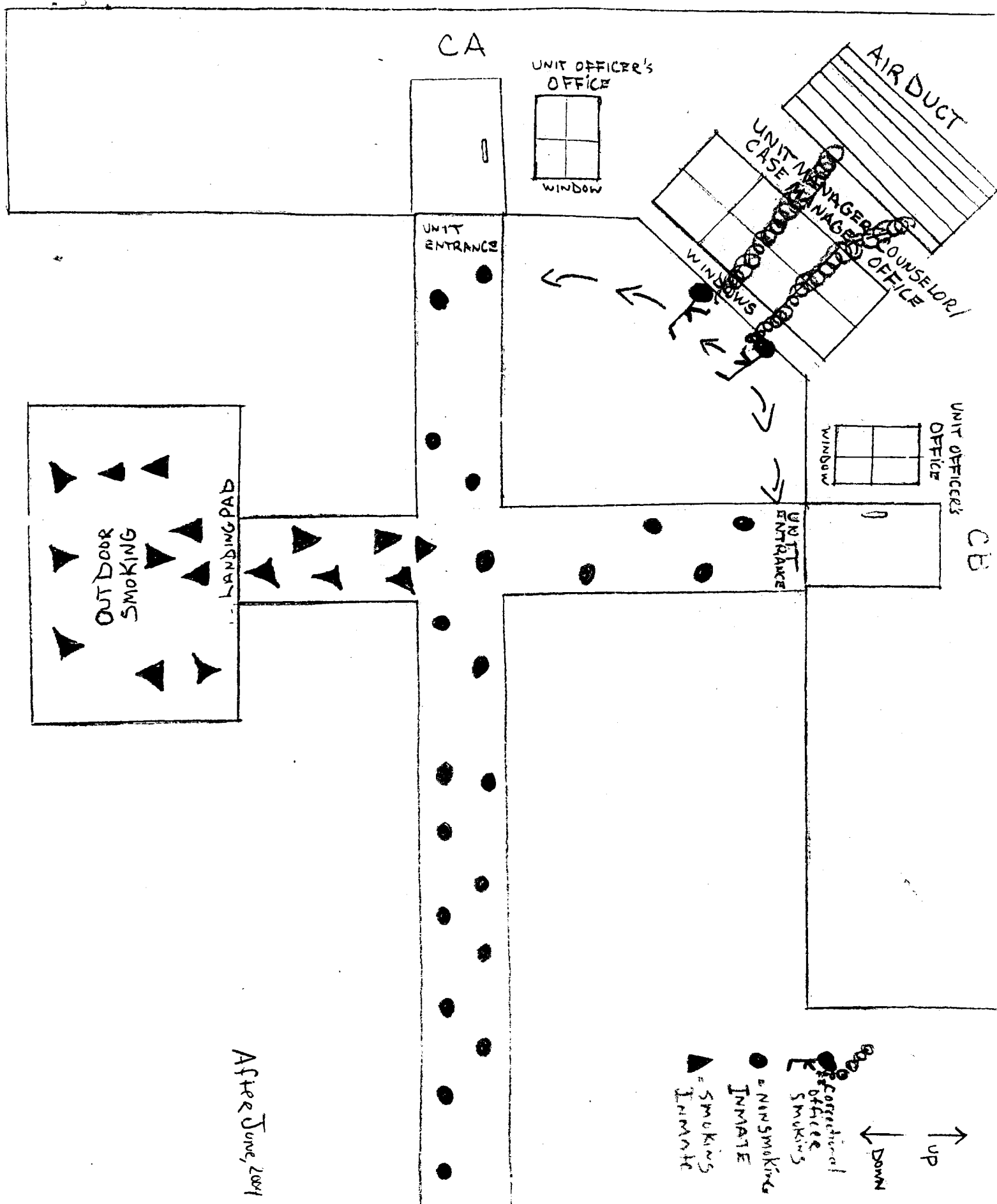
The results show patients can benefit from early detection, says Jeffrey H. Peters, surgery-department chairman at the University of Rochester Medical Center and a study co-author. "I believe we should be screening high-risk populations," says Dr. Peters. "The principle is clear—the earlier you pick up a cancer the more likely you are to cure it. There's very little debate about that."

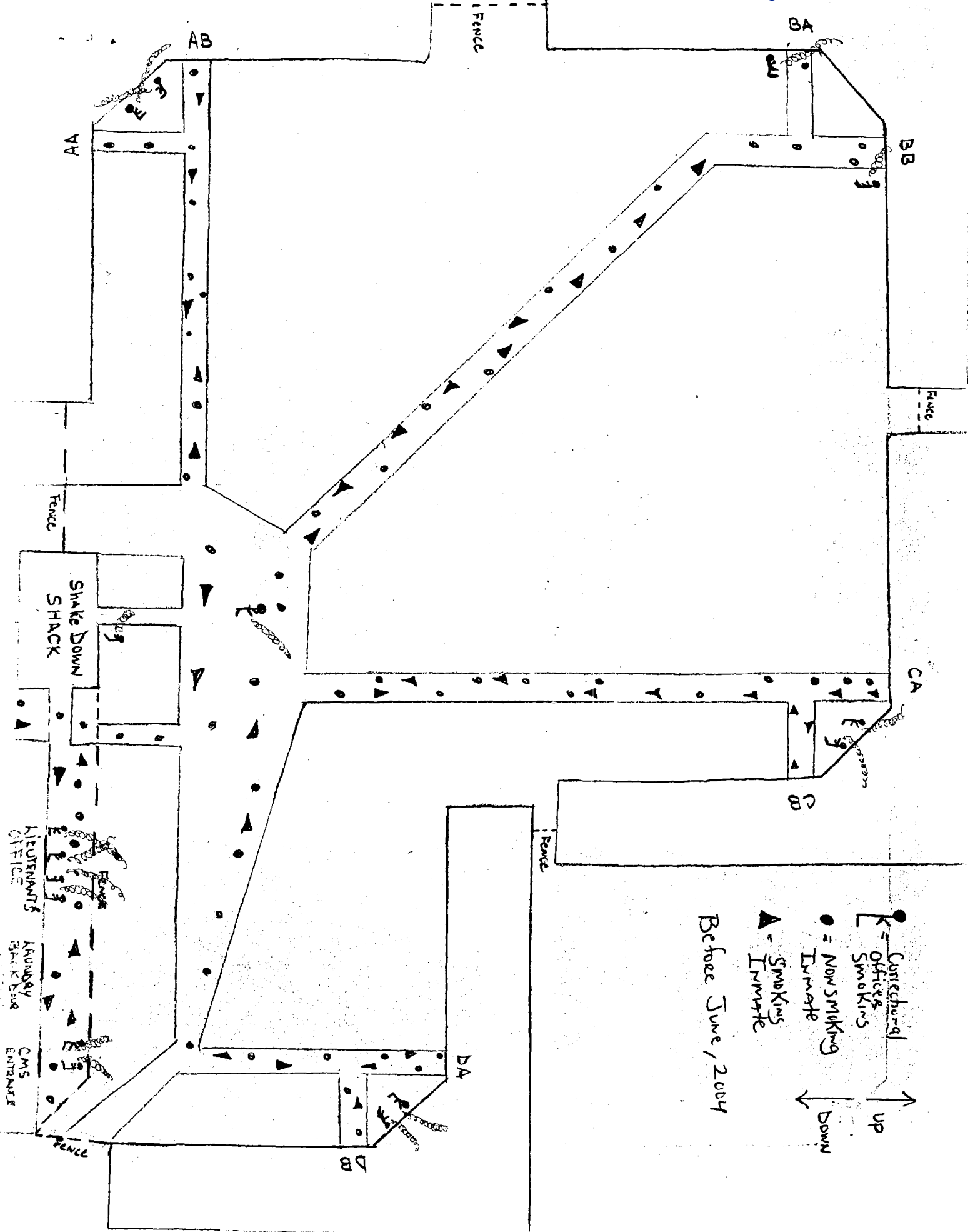
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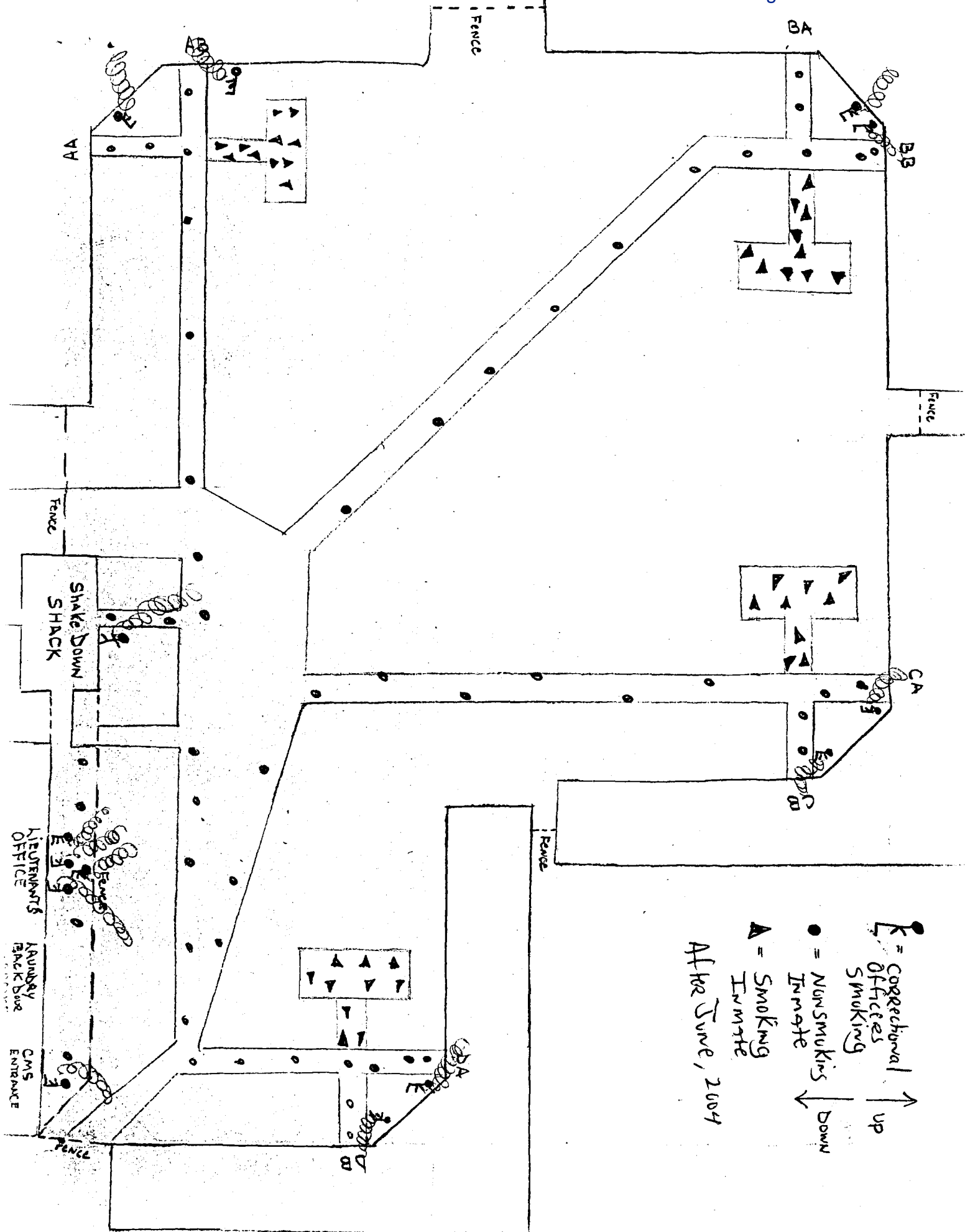
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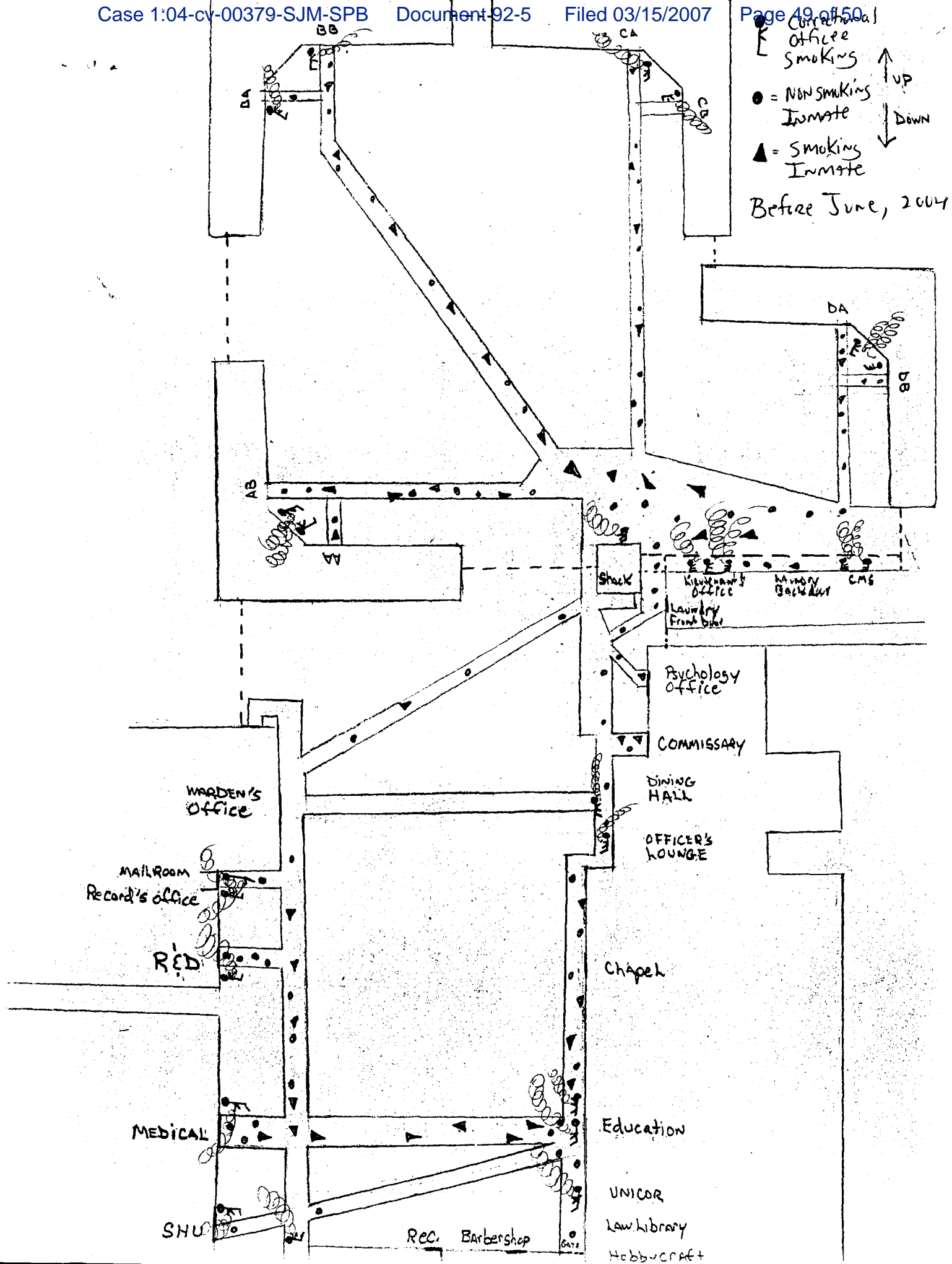


Before June 2, 2001









Correctional
Officer
Smoking
UP
DOWN
● = Non-Smoking Inmate
▲ = Smoking Inmate
After June, 2004

